

CLAIMS

5 1. An isolated polypeptide comprising an amino acid sequence having at least 80% sequence identity to the sequence of SEQ ID NOS:2, 4, 6, 8, 10, 12, 14 or 16.

2. The polypeptide of claim 1, wherein said polypeptide is an active AAP polypeptide.

10 3. The polypeptide of claim 2, wherein said amino acid sequence has at least 90% sequence identity to the sequence of SEQ ID NOS:2, 4, 6, 8, 10, 12, 14 or 16.

15 4. The polypeptide of claim 2, wherein said amino acid sequence has at least 98% sequence identity to the sequence of SEQ ID NOS:2, 4, 6, 8, 10, 12, 14 or 16.

5. An isolated polynucleotide encoding the polypeptide of claim 1, or a complement of said polynucleotide.

20 6. An isolated polynucleotide comprising a nucleotide sequence having at least 80% sequence identity to the sequence of SEQ ID NOS:1, 3, 5, 7, 9, 11, 13 or 15, or a complement of said polynucleotide.

25 7. The polynucleotide of claim 6, wherein said nucleotide sequence has at least 90% sequence identity to the sequence of NOS:1, 3, 5, 7, 9, 11, 13 or 15, or a complement of said polynucleotide.

8. The polynucleotide of claim 6, wherein said nucleotide sequence has at least 98% sequence identity to the sequence of SEQ ID NOS:1, 3, 5, 7, 9, 11, 13 or 15, or a complement of said polynucleotide.

30 9. An antibody that specifically binds to the polypeptide of claim 1.

10. A method of modulating angiogenesis comprising modulating the activity of at least one AAP.

5 11. The method of claim 10 wherein said modulating angiogenesis is increasing angiogenesis, and said modulating the activity comprises increasing the activity of at least one polypeptide selected from the group consisting of KLP, hBAZF, hTRG, hMX1, hMX2, hEF-G, and hMP.

10 12. The method of claim 10 wherein said modulating angiogenesis is decreasing angiogenesis, and said modulating the activity comprises increasing the activity of at least one polypeptide, wherein said at least one polypeptide comprises NHR.

15 13. The method of claim 10 wherein said modulating angiogenesis is decreasing angiogenesis, and said modulating the activity comprises decreasing the activity of at least one polypeptide selected from the group consisting of KLP, hBAZF, hTRG, hMX1, hMX2, hEF-G, and hMP.

20 14. The method of claim 10 wherein said modulating angiogenesis is increasing angiogenesis, and said modulating the activity comprises decreasing the activity of at least one polypeptide, wherein said at least one polypeptide comprises NHR.

25 15. The method of claim 11 wherein said increasing activity comprises increasing the expression of said at least one polypeptide.

30 16. The method of claim 13 wherein said decreasing activity comprises decreasing the expression of said at least one polypeptide.

17. The method of claim 15 wherein said increasing expression comprises transforming a cell to increase expression of a polynucleotide encoding said at least one polypeptide.

18. The method of claim 16 wherein said decreasing expression comprises transforming a cell to express a polynucleotide anti-sense to at least a portion of an endogenous polynucleotide encoding said at least one polypeptide.

5 19. The method of claim 13 wherein said decreasing activity comprises transforming a cell to express an aptamer to said at least one polypeptide.

10 20. The method of claim 13 wherein said decreasing activity comprises introducing into a cell an aptamer to said at least one polypeptide.

21. The method claim 13 wherein said decreasing activity comprises administering to a cell an antibody that selectively binds to said at least one polypeptide.

15 22. A method of treating tumors comprising decreasing angiogenesis by the method of claim 12.

23. A method of treating cancer comprising treating a cancerous tumor by the method of claim 22.

20 24. A method of treating myocardial infarction comprising increasing angiogenesis by the method of claim 11.

25 25. A method of promoting healing comprising increasing angiogenesis by the method of claim 11.

26. A method for determining whether a compound up-regulates or down-regulates the transcription of an AAP gene, comprising:
contacting said compound with a composition comprising a RNA polymerase and said gene and measuring the amount of said AAP gene transcription.

30 27. The method of claim 26, wherein said composition is in a cell.

28. A method for determining whether a compound up-regulates or down-regulates the translation of an AAP gene, comprising:

contacting said compound with a composition comprising a ribosome and a polynucleotide corresponding to a mRNA of said gene and measuring the amount of said AAP gene translation.

29. The method of claim 28, wherein said composition is in a cell.

30. A vector, comprising the polynucleotide of claim 5.

31. A cell, comprising the vector of claim 30.

32. A method of screening a tissue sample for tumorigenic potential, comprising:
measuring expression of at least one AAP gene in said tissue sample.

33. The method of claim 32, wherein said measuring is measuring an amount of a polypeptide encoded by said at least one AAP gene.

34. The method of claim 32, wherein said measuring expression is measuring an amount of mRNA corresponding to said at least one AAP gene.

35. A transgenic non-human animal, having at least one disrupted AAP gene.

36. The transgenic non-human animal of claim 35, wherein the non-human animal is a mouse.

37. A transgenic non-human animal, comprising an exogenous polynucleotide having at least 80% sequence identity to the sequence of SEQ ID NOS:1, 3, 5, 7, 9, 11, 13 or 15, or a complement of said polynucleotide.

38. The transgenic non-human animal of claim 37, wherein said exogenous polynucleotide has at least 90% sequence identity to the sequence of SEQ ID NOS:1, 3, 5, 7, 9, 11, 13 or 15 , or a complement of said polynucleotide.

5 39. The transgenic non-human animal of claim 37, wherein said exogenous polynucleotide has at least 98% sequence identity to the sequence of SEQ ID NOS:1, 3, 5, 7, 9, 11, 13 or 15 , or a complement of said polynucleotide.

10 40. A method of screening a sample for an AAP gene mutation, comprising:
comparing an AAP nucleotide sequence in the sample with SEQ ID NOS:1, 3, 5, 7, 9, 11, 13 or 15 .

15 41. A method of determining the clinical stage of tumor comprising comparing expression of at least one AAP gene in a sample with expression of said at least one gene in control samples.